



Letter to the Editor

Dear Editor

Eosinophilic pleural effusion due to gliclazide

A 52 year-old male ex-smoker was admitted to our hospital complaining of pleuritic pain in the right chest. The patient had had normal findings on a previous chest roentgenogram, with no previous pulmonary history or occupational exposure. He had been suffering from diabetes mellitus type II controlled by diet since 1990. Two weeks before admission gliclazide was administered. On admission, a chest X-ray revealed ipsilateral moderate pleural effusion and small lung infiltrates of the right side, confirmed by computed tomography scan of the chest. The leukocyte count was $7500 \text{ cells ml}^{-1}$ with 20% eosinophils, the hematocrit was 44% and the erythrocyte sedimentation rate 14 mm for the first hour. A PPD skin test (Mantoux) with 5 IU PPD was negative. The differential cell counts of exudative pleural fluid showed 8% neutrophils, 12% lymphocytes and 80% eosinophils. Sputum cultures were negative for *Mycobacterium tuberculosis*. Pleural biopsy was negative for tuberculosis or malignancy. Ova and parasites were not found in the stools. Fiberoptic bronchoscopy was performed with normal endoscopic findings. The patient refused transbronchial biopsy. Bronchial brushing and washing specimens were negative for acid-fast bacilli, fungi and malignant cells. A bronchoalveolar lavage showed 78% macrophages, 10% neutrophils, 10% lymphocytes and 2% eosinophils. Gliclazide was discontinued and 1 month later the patient became asymptomatic, eosinophil counts in the blood were within normal range and the chest roentgenogram showed complete resolution of the pleural effusion.

Eosinophilic pleural effusions, defined as 10% or more eosinophils, account for between 5–8% of exudative pleural effusions (1) and imply a favourable prognosis, based on their association with benign diagnoses (2). The gliclazide is

an oral sulfonylurea hypoglycaemic agent, which stimulates insulin secretion by β -cells of the pancreas. Sulfonylurea compounds such as chlorpropamide (3,4) or tolazamide (5) have been reported to cause eosinophilic parenchymal lung disease. However, our patient presented with sub-acute pleural illness with minimal pulmonary infiltrates. Although, gliclazide has been reported to cause blood eosinophilia (unpublished data from pharmaceutical company's database), there is no report in the literature of eosinophilic pleural effusion due to sulfonylurea compounds. However, the rapid clinical and roentgenographic improvement and the return to normal of the blood eosinophilia after discontinuation of the gliclazide, strongly suggest a drug-induced disease.

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